

Divergent Phenotypes and Persistent Disability: A Comparative Cohort Study of Arterial Ischaemic and Haemorrhagic Stroke in Chinese Paediatric Patients

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Abstract: Background: Paediatric stroke poses a significant clinical challenge with lifelong disability risks. However, comparative analyses delineating subtype-specific manifestations and outcomes between arterial ischemic stroke (AIS) and haemorrhagic stroke (HS) are lacking.

Objective: To characterize and compare the AIS and HS subtypes in a paediatric cohort, focusing on clinical presentation, aetiology, lesion distribution, and outcomes.

Methods: This retrospective cohort study included consecutive paediatric patients (1 month-18 years) with first-ever AIS or HS admitted to Beijing Tiantan Hospital between June 2019 and May 2024. Cases were identified through structured electronic medical record review using standardized diagnostic criteria.

Results: The cohort comprised 72 paediatric stroke cases (42 AIS, 30 HS) with comparable age distributions (median 8.0 vs 8.5 years) but distinct clinical profiles. While AIS patients predominantly presented with focal deficits (73.8% hemiparesis vs 30.0% in HS), HS cases more frequently exhibited diffuse symptoms, including headache (56.7% vs 11.9%) and nausea/vomiting (36.7% vs 4.8%). Etiological evaluation revealed striking differences: arteriopathies (particularly moyamoya disease and vasculitis) were dominant in AIS, whereas structural vascular anomalies (arteriovenous malformations [AVMs] and cavernomas) were characteristic of HS. At the 12-month follow-up, persistent neurological sequelae were common in both groups (AIS 67.6%, HS 64.3%), though disability patterns diverged; AIS survivors demonstrated predominantly motor deficits (56.8% vs 28.6%) while cognitive impairment was more prevalent in HS (21.4% vs 13.5%). Mortality rates remained low (AIS 2.7%, HS 7.1%), with recurrence observed exclusively in AIS cases (5.4%).

Conclusion: Acute paediatric AIS and HS exhibit distinct clinical phenotypes—focal neurological deficits are predominant in AIS versus diffuse symptoms (headache/nausea) in HS, enabling early syndromic differentiation. Despite treatment, persistent neurological morbidity affects >64% of survivors, with subtype-specific disability patterns: AIS is primarily associated with motor impairments, while HS demonstrates greater cognitive sequelae. The low mortality

(<7.1%) and recurrence rates reflect advancements in care, yet the enduring disability burden underscores the necessity for subtype-tailored rehabilitation and systematic long-term surveillance.

Keywords: Paediatric stroke, Arterial ischaemic stroke, Haemorrhagic stroke, Clinical phenotypes, Aetiology, Neurological outcomes

1. Introduction

Paediatric stroke represents a significant cause of acquired childhood disability, with an annual incidence of 2-13 per 100,000 children ^[1,2]. Distinct from adult stroke, paediatric cases demonstrate unique pathophysiology requiring age-specific management strategies ^[3,4]. Although arterial ischemic stroke (AIS) and haemorrhagic stroke (HS) constitute over 80% of cases ^[5], their comparative profiles remain inadequately characterized, particularly in Asian populations.

Despite advancements in neuroimaging, diagnostic delays persist due to nonspecific presentations and a lack of standardized paediatric protocols ^[6]. Aetiologically, AIS is predominantly associated with arteriopathies (e.g., moyamoya disease), while HS principally results from vascular malformations ^[7]. However, examinations of regional variations and direct clinico-radiological comparisons within unified cohorts are notably absent.

To this end, this study systematically compared 72 consecutive cases (42 AIS, 30 HS) to: (1) delineate subtype-specific phenotypic profiles, and (2) characterize differential long-term outcomes. Our findings establish an evidence-based framework for precision medicine in paediatric stroke care.

2. Methods

2.1 Study Population

This retrospective cohort study comprised consecutive paediatric stroke patients admitted to the Department of Paediatrics at Beijing Tiantan Hospital between June 2019 and May 2024. Participants were classified into an AIS group and a HS group based on: (1) neuroimaging evidence (magnetic resonance imaging [MRI]/computed tomography [CT]) reviewed by two blinded neuroradiologists, and (2) clinical criteria. AIS required acute focal neurological deficits lasting >24 hours with radiologically confirmed infarction, while HS required equivalent deficits with haemorrhage confirmation. Neonates (< 44 weeks corrected gestational age) and transient ischemic attacks were excluded.

2.2 Data Collection

Demographic (sex, age), clinical (motor/sensory/cognitive deficits, seizure history), and neuroimaging characteristics (lesion topography, vascular territories) were systematically extracted from the patients' electronic medical records. Aetiological subtyping was performed according to the International Paediatric

Stroke Study criteria ^[4]. Functional outcomes (residual deficits, recurrence) were assessed at 12 months post-discharge (median follow-up: 12.4 months; interquartile range [IQR]: 10.8-14.1 months) via structured telephone interviews.

2.3 Statistical Analysis

Analyses were performed using SPSS 26.0 (IBM). Normality was assessed with the Shapiro-Wilk test. Categorical variables were compared using Fisher's exact test; continuous variables with the Wilcoxon-Mann-Whitney test. Benjamini-Hochberg false discovery rate (FDR) correction was selected over Bonferroni adjustment to balance Type I/II errors. Effect sizes are reported as risk ratios (RR, incidence >10%) or odds ratios (OR) with 95% confidence intervals (CIs). Statistical significance was defined as $P_{adj} < 0.05$.

2.4 Ethical Considerations

The study was approved by the local ethics committee. Written informed consent was obtained from the guardians of all paediatric participants prior to their inclusion in the study, ensuring confidentiality and anonymity of the data collected.

3. RESULTS

3.1 Demographic Data

A total of 75 consecutive paediatric stroke patients were enrolled in the study. Three cases (two cerebral venous sinus thrombosis [CVST], one mixed stroke) were excluded from the comparative analyses due to insufficient subgroup sizes for statistical comparisons ($n < 5$ per subtype). The primary cohorts comprised arterial ischemic stroke (AIS, $n = 42$, 56.0%) and haemorrhagic stroke (HS, $n = 30$, 40.0%) patients (Table 1).

Both groups had similar baseline characteristics, including a male predominance (AIS: 69.0%; HS: 60.0%) and comparable age distributions (median [IQR]: AIS 8.0 [5.0-10.0] vs. HS 8.5 [6.3-11.0] years; $P = 0.32$). School-aged children (6–12 years) represented the largest subgroup in both the AIS (52.4%) and HS (56.7%) populations, followed by preschool children (1–5 years: AIS 28.6% vs. HS 26.7%) (Table 1).

The two CVST cases (a 13-year-old and a 10-year-old male) and one mixed stroke case (a 6-year-old male with moyamoya disease) demonstrated distinct clinical profiles (Supplementary **Table 1**).

Table 1. Demographic Characteristics of AIS and HS Cohorts ($n = 72$)

Characteristic	AIS ($n=42$)	HS ($n=30$)	P value
Male gender	29 (69.0%)	18 (60.0%)	0.430
Age, years			0.320*
Median [IQR]	8 [5.0-10.0]	8.5 [6.3-11.0]	
<1 year	2 (4.8%)	0 (0.0%)	0.500

1–5 years	12 (28.6%)	8 (26.7%)	1.000
6–12 years	22 (52.4%)	17 (56.7%)	0.820
13–18 years	6 (14.3%)	5 (16.7%)	1.000

AIS, arterial ischemic stroke; HS, haemorrhagic stroke. *Mann-Whitney U test ($U = 542$), all others by χ^2 test. †Three excluded cases (2 CVST, 1 mixed) not tabulated.

Supplementary Table S1. Clinical characteristics of excluded CVST and mixed stroke cases

Parameters	CVST Case 1	CVST Case 2	Mixed Stroke Case
Age (years)	13	10	6
Sex	Male	Male	Male
Aetiology	Hyperhomocysteinemia	Postinfectious	Moyamoya disease
Clinical presentation	Headache, hemiparesis, limb weakness	Headache, nausea/vomiting, dizziness, gait instability	Headache, somnolence
Neuroimaging findings	Superior sagittal sinus (SSS) thrombosis	Extensive thrombosis (SSS, straight sinus, transverse/sigmoid sinuses)	Bilateral hemispheric infarcts with occipitoparietal haemorrhages
Treatment	Low-molecular-weight heparin (LMWH) to warfarin	LMWH	Encephaloduroarteriosynangiosis (EDAS) surgery
Outcome	Full recovery	Death	Cognitive impairment, paralysis, epilepsy

CVST, cerebral venous sinus thrombosis; EDAS, encephaloduroarteriosynangiosis; LMWH, low-molecular-weight heparin.

Clinical Characteristics

The AIS and HS cohorts demonstrated significantly distinct neurological presentations. Focal deficits were predominant in AIS, with hemiparesis (73.8% vs. 30.0%; $P_{\text{adj}} = 0.001$, $RR = 2.46$ [95% CI, 1.36–4.44]), facial palsy (14.3% vs. 0.0%), and dysarthria (11.9% vs. 6.7%) being most characteristic. In contrast, HS patients predominantly exhibited diffuse symptoms, including headache (56.7% vs. 11.9%; $P_{\text{adj}} < 0.001$), nausea/vomiting (36.7% vs. 4.8%; $P_{\text{adj}} = 0.002$), and decreased consciousness (33.3% vs. 9.5%). These profiles suggest that hemiparesis should prompt evaluation for AIS, whereas headache with nausea/vomiting should raise suspicion for HS (**Table 2**).

Table 2. Comparative clinical features of paediatric arterial ischaemic and haemorrhagic stroke

Clinical feature	AIS (n=42)	HS (n=30)	Crude P value†	$P_{\text{adj}}^{\ddagger}$
Focal signs				
Hemiparesis	31 (73.8)	9 (30.0)	<0.001	0.001
Facial palsy	6 (14.3)	0 (0.0)	0.029	0.290
Dysarthria	5 (11.9)	2 (6.7)	0.693	1.000
Ataxia	5 (11.9)	4 (13.3)	1.000	1.000
Visual disturbance	0 (0.0)	2 (6.7)	0.173	1.000
Numbness	6 (14.3)	1 (3.3)	0.236	1.000
Diffuse signs				
Decreased consciousness	4 (9.5)	10 (33.3)	0.017	0.170
Seizures	6 (14.3)	5 (16.7)	0.757	1.000
Headache	5 (11.9)	17 (56.7)	<0.001	<0.001
Nausea/Vomiting	2 (4.8)	11 (36.7)	<0.001	0.002

AIS, arterial ischemic stroke; HS, haemorrhagic stroke. †Fisher's exact test. ‡FDR-adjusted P value ($q = 0.10$).

Aetiologies and Risk Factors

Comprehensive evaluation identified distinct aetiologic patterns between the stroke subtypes, with causative factors detected in 88.1% (37/42) of AIS and 93.3% (28/30) of HS cases. The AIS cohort demonstrated predominant arteriopathies, particularly moyamoya disease (21.4% vs. 0% in HS, $P_{adj} = 0.018$) and immune-mediated vasculitis (19.0% vs. 3.3%, $P_{adj} = 0.066$), whereas HS was characterized by structural vascular anomalies, including cerebral cavernous malformations (33.3% vs. 4.8%, $P_{adj} < 0.001$; OR = 9.80 [95% CI, 2.01-47.7]) and arteriovenous malformations (26.7% vs. 0%, $P_{adj} < 0.001$). Notably, only two cardiac-related stroke cases were identified, both in the AIS cohort. The complete spectrum of risk factors for both stroke subtypes is presented in **Table 3**.

Lesion Distribution Patterns

In the AIS cohort, lesions most frequently involved the basal ganglia (19/42, 45.2%) and supratentorial cortical regions (13/42, 31.0%), particularly the frontal and parietal lobes, with multifocal distribution observed in 23.8% (10/42) of cases. Vascular territory analysis revealed a predominance of anterior circulation involvement (69.0%, 29/42), significantly exceeding posterior (16.7%, 7/42) and combined circulations (14.3%, 6/42). In contrast, HS lesions were primarily localized to the basal ganglia (20.0%, 6/30), temporoparietal junctions (16.7%, 5/30), and brainstem (16.7%, 5/30).

Table 3. Complete aetiologic distribution of paediatric stroke by subtype

Aetiologic category	AIS (n=42)	HS (n=30)	Crude P value	$P_{adj}^{\dagger}$
Arteriopathy				
Cerebral cavernous malformation	2 (4.8)	10 (33.3)	<0.001	<0.001
Arteriovenous malformation	0 (0.0)	8 (26.7)	<0.001	<0.001
Moyamoya disease	9 (21.4)	0 (0.0)	0.003	0.018
Immune-mediated vasculitis	8 (19.0)	1 (3.3)	0.022	0.066
Aneurysms/Structural abnormalities	5 (11.9)	3 (10.0)	1.000	1.000
Haematologic/thrombophilia				
- Prothrombotic states	2 (4.8)*	0 (0.0)	0.257	0.514
- Haemolytic anaemia	1 (2.4)	0 (0.0)	1.000	1.000
Infection	2 (4.8)	0 (0.0)	0.507	0.761
Cardiac disease	2 (4.8)	0 (0.0)	0.507	0.761
Tumour-related	2 (4.8)	4 (13.3)	0.235	0.564
Trauma/Toxic exposure	4 (9.5)	2 (6.7)	0.707	0.848
Undetermined	5 (11.9)	2 (6.7)	0.688	0.826

† FDR-adjusted P value ($q = 0.05$). *Prothrombotic states include hyperlipidaemia ($n = 1$) and hyperhomocysteinemia ($n = 1$).

Clinical Outcomes

During a median follow-up of 12.4 months (IQR 10.8-14.1), mortality rates differed between the cohorts: one AIS death (2.7%) resulted from moyamoya disease progression, whereas two HS deaths (7.1%) occurred due to brain herniation ($n = 1$, at 3 months) and intracranial tumour progression ($n = 1$, at 5 months).

Disease recurrence occurred only in the AIS cohort (5.4%; $n = 1$ moyamoya progression and $n = 1$ vasculitis recurrence), with no HS rebleeding episodes observed. Persistent neurological sequelae occurred in 67.6% of AIS and 64.3% of HS survivors, with AIS showing a higher prevalence of motor deficits (56.8% vs. 28.6%, $P = 0.045$) (Table 4).

Table 4. Outcomes of paediatric stroke survivors

Outcome measures	AIS (n=37)	HS (n=28)	Crude P value†	Padj‡	Effect size (95% CI)
Main Outcomes					
Complete recovery	11 (29.7)	8 (28.6)	0.902	0.902	RR 1.04 (0.50–2.15)
Death	1 (2.7)	2 (7.1)	0.586	0.586	OR 0.37 (0.03–4.03)
Persistent disability	25 (67.6)	18 (64.3)	0.764	0.764	RR 1.05 (0.73–1.51)
Neurologic Sequelae					
Motor deficits	21 (56.8)	8 (28.6)	0.045	0.100	RR 1.99 (1.03–3.86)
Cognitive impairment	5 (13.5)	6 (21.4)	0.744	0.744	RR 0.63 (0.21–1.88)
Epilepsy	6 (16.2)	3 (10.7)	1.000	1.000	RR 1.51 (0.41–5.53)
Language disorders	4 (10.8)	1 (3.6)	0.772	0.772	RR 3.03 (0.36–25.74)
Recurrence	2 (5.4)	0 (0.0)	1.000	1.000	-

†Fisher's exact test. ‡FDR-adjusted P value ($q = 0.10$).

DISCUSSION

AIS and HS represent the predominant subtypes of paediatric stroke, accounting for approximately 80% of cases [2]. Our study revealed a predominance of ischemic stroke (58.3%) over haemorrhagic (41.7%) among paediatric stroke cases, consistent with established epidemiological literature generally reporting ischemic pathology in nearly two-thirds of childhood strokes [8,9]. The median onset age (AIS: 8.0 years; HS: 8.5 years) and peak incidence among school-aged children (6–12 years) align with meta-analytic data [10]. Both subtypes showed male predominance (AIS: 69.0%; HS: 60.0%), consistent with prior studies [11], though the exact mechanisms—potentially involving sex-linked endothelial modulation or behavioural risk factors—remain incompletely elucidated [12,13].

Clinically, AIS manifested predominantly with focal neurological deficits, particularly hemiparesis (73.8%), whereas HS presentations were characterized by diffuse symptoms such as headache (56.7%) and impaired consciousness (33.3%), likely reflecting mass effects or acute intracranial hypertension [3]. Seizure incidence was comparable between the groups (AIS: 14.3%; HS: 16.7%), aligning with existing literature [15,16].

Our findings identified non-atherosclerotic arteriopathies as the primary aetiology of paediatric AIS. Moyamoya disease represented 21.4% of AIS cases, consistent with its higher prevalence in Asian populations [17]. The exclusively ischaemic presentation of moyamoya disease in our cohort supports its characteristic pathophysiology of progressive carotid occlusion leading to collateral formation rather than haemorrhage [18]. Immune-mediated vasculitis was identified in 19.0% of AIS cases, aligning with literature reporting stroke occurrence in 15–30% of such patients, predominantly ischemic in nature (75–85%) [19]. Cardiac

aetiologies were less frequent in our AIS cohort (4.8%) compared to Western cohorts (25–36%)^[20], potentially reflecting regional disparities in congenital heart disease prevalence or diagnostic practices. Our study identified additional AIS risk factors encompassing infections, metabolic disorders, and cerebral infarction following wasp envenomation. For suspected AIS, current guidelines recommend: (1) thrombophilia screening (coagulation studies, protein C/S activity, antiphospholipid antibodies); (2) metabolic workup (fasting homocysteine, lipid panel); and (3) cardiac evaluation (echocardiography, electrocardiogram)^[21].

In HS patients, structural vascular anomalies were predominant, with cerebral cavernous malformations (CCMs) and arteriovenous malformations (AVMs) collectively accounting for 60% of cases; this distribution is consistent with the paediatric HS literature^[7]. Approximately 10% of cases remained aetiologically undefined despite acute-phase evaluation, comparable to published rates; this is potentially attributable to occult vascular abnormalities undetectable by conventional imaging^[3].

The mortality rates in our cohort (AIS: 2.7%; HS: 7.1%) demonstrate marked improvement compared to historical data (HS: 25–54%)^[22,23], likely reflecting advances in neurocritical care and neurosurgical interventions. However, persistent neurological sequelae remained common (AIS: 67.6%; HS: 64.3%), with distinct disability profiles: motor deficits were predominant in AIS survivors, while HS patients showed greater neurocognitive impairments. These findings are consistent with international cohorts^[23] and highlight the need for subtype-specific rehabilitation approaches.

The AIS cohort exhibited a 4.8% recurrence rate (2/42), lower than the 10% reported in population-based studies^[24]. This possibly reflects comprehensive tertiary-centre management, including aetiology-specific secondary prevention and timely surgical/endovascular therapies^[25].

Strengths and Limitations

This study offers a detailed, imaging-validated comparison of paediatric AIS and HS, employing standardized classifications and rigorous statistical adjustments to delineate subtype-specific patterns in presentation, aetiology, and outcomes. As a single-centre investigation, the findings may reflect regional referral biases and institutional protocols, limiting the generalizability of the findings to resource-limited settings. The median 12.4-month follow-up enabled short-term outcome assessment but precluded evaluation of long-term neurodevelopmental sequelae. This is critical given paediatric stroke's lifelong disability risks.

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